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ABSTRACT

The research described in this article addressed the question of why siblings commonly have different developmental outcomes despite their common beginnings. The studies analyzed behavioral development, especially through examination of deviant behaviors and intellectual development, by tracing siblings' different life histories. The work is based upon a theoretical framework that attributes behavioral variation to causal sources that define composite variance components. These variance components are composite because a variety of specific influences can unite to produce variation of a particular type. The article is divided into three sections that review the research program and findings. The first section focuses on using sibling and twin studies to attribute behavioral variation to composite variance components used in behavior genetic research. The second section discusses the issue of comorbidity--traits that are related in their familial co-occurrence. The third section describes specific sources of composite variance components. The chapter closes with a look at unanswered questions and directions for future work. Contains 37 references. (SD)

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Delinquency and IQ: Using Siblings to Find Sources of Variation

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In any family, siblings may lead different lives. One child may study hard at school and attain high grades and later a good paying job; another child may fail in school and later find unskilled work. In the Wideman family, John and his younger brother Robby both lived in an urban community and were raised in relative poverty. John, however, won a scholarship to the University of Pennsylvania, where he completed both his undergraduate and graduate studies (he then became a professor of English). In contrast, Robby spent his adolescence stealing to buy drugs; eventually, he was arrested and convicted as an accessory to murder. In his autobiographical book *Brothers and Keepers* (Wideman, 1984), John tried to understand their different life courses. According to John, their mother had always noticed differences between him and Robby, "The bad seed, the good seed. Mommy's been saying for as long as I can remember: That Robby . . . he wakes up in the morning looking for the party . . . Where's it at today? What's it gonna be today? Where's the fun?" (p. 20). Although few siblings have lives as divergent as John and Robby Wideman, they commonly have

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different developmental outcomes. To analyze behavioral development scientifically, we favor tracing siblings' different life histories.

This chapter describes our general research programs and a number of specific research projects focusing on adolescent development. We investigated various behavioral traits, primarily "deviant" behaviors (e.g., delinquency) and intellectual development. In these research projects, we made considerable use of the sibling research design, namely, sampling more than one child per recruited family. Our interest in siblings is related to theoretical concerns with the etiology of behavioral variation. First, we attribute behavioral variation to particular causal sources that define composite variance components. These variance components are *composite* because a variety of specific influences can unite to produce variation of a particular type, for instance, genes with many different physiological effects may contribute to a trait's heritability. Questions asked about composite variance components include: Is a trait heritable (e.g., Do genes cause variation in the trait?)? Is it influenced by shared family environment (e.g., Do environmental influences cause siblings to be similar?)? Is there a social influence of siblings on each other (e.g., Do specific elements in the social environment influence development?)? Second, we move beyond composite variance components to the way in which they operate on behavioral development—to Anastasi's (1958) question "how?" That is, composite variance components can be seen as opportunities to explore developmental process further if they can be attributed to specific influences and causal pathways.

This chapter is divided into three sections that review our research program and related findings. The first section focuses on using sibling and twin studies to attribute variation in behavior to these composite variance components used in behavior genetic research. The second section discusses the issue of *comorbidity*—traits that are related in their familial co-occurrence. The third section describes specific sources of composite variance components. The chapter closes with a look at unanswered questions and directions for future work. This chapter explores both methodology—what can be done with different kinds of sibling data—and content—what we have learned about the origin of variation in IQ and delinquency.

COMPOSITE VARIANCE COMPONENTS

This chapter describes sibling studies of intelligence (IQ) and delinquent behavior. Human intelligence refers to abstract reasoning and problem-solving abilities as assessed by standardized IQ tests. Intelligence can be regarded as a "fuzzy" concept—no simple definition adequately captures all its facets (e.g., Sternberg & Detterman, 1986). Nonetheless, it is generally agreed that IQ is closely related to success in mastering school-taught academic subjects. The origin of individual differences in IQ has been a perennial research topic in the social sciences.

Delinquent behaviors include acts commonly forbidden by law such as burglary, theft using violence or the threat of force, deliberately injuring another person, and so on. Although specific delinquent acts vary in their legal status across cultures and historical periods, social norms and formal sanctions against them are nearly cross-cultural universals. Levels of delinquency tend to rise from early adolescence into early adulthood (age 25 or less) and then fall sharply later in the life course. Both in the United States and in all societies with extensive records, more males than females are delinquent—especially in those acts that are overtly aggressive. Finally, within each sex, the delinquency distribution is positively skewed across individuals: Most adolescents commit few or no delinquent acts, but a minority are frequently and persistently delinquent.

This latter, individual variation in delinquent behavior is one focus of developmental research. Behavior genetic research designs can apportion behavioral variation to composite variance components. First, they can apportion variation between familial and nonfamilial determinants. Second, trait variation can be further attributed to heredity or shared or nonshared environmental variance components. Heritable trait variation results from genetic effects, that is, from the influence of the substitution of one gene for another relevant to a trait (e.g., the substitution of gene B for A can change blood type AA to blood type AB). Shared environmental influences, by definition, refer to all environmental experiences operating to make siblings alike but different from children in other families; they are environmental influences conceptually tied to the family unit. Finally, nonshared en-

vironmental influences operate on each person uniquely; they make reared-together siblings dissimilar in behavior.

Are IQ and Delinquency Familial?

To discover genetic or shared (family) environmental influences, an initial question must be whether a trait "runs" in families. In the case of siblings, the question is: If one sibling exhibits a trait, is it also more likely to appear in another sibling? The simplest way to answer this question is merely to compute either sibling correlations or concordances (e.g., risk to an unselected sibling, given that a selected sibling is affected). In many studies of official delinquency, evidence has been obtained that delinquent behavior is "familial" (Ellis, 1982).

In our research program, we extended this basic sibling research design to examine several siblings who differ in birth order simultaneously (Rodgers & Rowe, 1985; Rowe, Rodgers, & Meseck-Bushey, 1992). If genetic influences—not environmental influences—were primarily responsible for trait variation, then birth order should be relatively unimportant because the recombination of maternal and paternal genes ignores birth order in the production of offsprings' traits. That is, a first-born child's behavioral resemblance to a second-born child should about equal that of his or her resemblance to a third-, forth-, or fifth-born child. However, many social influence factors may influence birth-order adjacent children to be more like one another than birth-order distant children, for example, if parents treat children close in birth order more alike because they are more similar in age. Sibling data never unambiguously separate genetic and family environmental influences, but equal sibling correlations across birth orders would be unexpected in some environmental explanations for behavioral variation.

Table 7.1 presents sibling correlations across four birth orders for both IQ and self-reported delinquency (Rodgers & Rowe, 1985; Rowe, Rodgers, & Meseck-Bushey, 1992). For both developmental outcomes, the sibling correlations are remarkably uniform across birth orders; in particular, more separated children are not less alike in their behavioral traits. The mean correlation for IQ (r = .57) was greater than that for self-reported delinquency (r = .35), indicating greater shared family influence on the former than on the latter. In both studies, regression

TABLE 7.1 SIBLING CORRELATIONS FOR IQ AND SELF-REPORT DELINQUENCY IN FOUR CHILDREN FAMILIES

IQ Matrix[a]

	Birth Order of Child			
Birth Order	1	2	3	4
1	1.0			
2	.62	1.0		
3	.55	.57	1.0	
4	.58	.57	.55	1.0

Self-Report Delinquency Matrix[b]

Birth Order	1	2	3	4
1	1.0			
2	.19	1.0		
3	.49	.32	1.0	
4	.45	.32	.34	1.0

[a]From "Does Contiguity Breed Similarity? A Within-Family Analysis of Nonshared Sources of IQ Differences between Siblings," by J.L. Rodgers & D.C. Rowe, 1985, *Developmental Psychology, 21*, p. 745. Copyright 1985 by the American Psychological Association. Reprinted with permission.

[b]From "Sibling Delinquency and the Family Environment: Shared and Unshared Influences," by D.C Rowe, J.L. Rodgers, & S. Meseck-Bushey, 1992, *Child Development, 63*, p. 63. Copyright 1992 by the Society For Research In Child Development. Reprinted with permission.

analyses were done to relate sibling similarity to family composition and age-spacing variables. Neither set of regressions suggested that these variables conditioned the siblings' behavioral resemblance. Given the large genetic component underlying IQ variation (Plomin, DeFries, & McClearn, 1990), the IQ results were not surprising. Given also the greater influence of environmental variation on delinquency (DiLalla & Gottesman, 1989), these findings were surprising. Later, we present a different approach showing that siblings' resemblance in self-reported delinquency can be conditioned on other variables.

The sibling correlations yield information about the relative magnitude of familial and nonfamilial influences on trait variation. The square of the mean correlation in Table 7.1 suggests that about 32% of IQ variation and 12% of delinquency variation in one sibling can be predicted from knowing the other's status. This interpretation would be followed in a practical intervention that, for instance, sought to use a low-IQ or highly delinquent sibling as a "risk factor" for younger siblings' behavior.

However, squaring the correlation to estimate the variance is incorrect for another application—estimating the total influence of familial sources of behavioral variation. In this case, the sibling correlation *is* variance explained.

This second interpretation of the sibling correlation is shown in Figure 7.1, in which z is the loading of an observed trait on its common factor of (unmeasured) causes. According to the rules of path analysis (Loehlin, 1987), the sibling correlation in Figure 7.1 equals z^2; that is, it is a product of the path coefficients connecting the siblings' phenotypes. By another path-analytic rule, variance explained in a manifest variable is the square of its loading on the common factor—once again, z^2. Thus, in this model, variance explained in the manifest variable equals the sibling correlation. Of course, this conclusion assumes that z is the same for younger and older siblings. In general, the older and younger siblings would represent, except for age, just two equivalent samples of children. Except for traits strongly associated with age, family effects on each child should be about equal. This assumption is reasonable for both IQ and delinquency in children during middle adolescence.

From the mean delinquency correlation in Table 7.1, one may say that 35% (r = .35) of delinquency variation is attributable to common familial influences. These familial effects would be even stronger if adjusted for measurement reliability. This adjustment is merely the sibling correlation divided by the reliability coefficient (r_s/r_{ii}). A reliability of about .75 for delinquency, for instance, would raise the estimated sibling correlation from .35 to .47, indicating that about half of delinquency variation can be attributed to common familial determinants. These common familial influences also may explain the year-to-year stability of delinquency in adolescence. In a path-analytic model, familial influences contributing to sibling resemblance at any one measurement time also contributes to delinquency's *within* person correlation between times of measurement (Rowe & Britt, 1991).

The sibling correlation also yields information relevant to the etiology of sex differences. For IQ, same- and opposite-sex sibling correlations are about equal, indicating that the shared familial influences on female and male IQs are identical. In contrast, in two-child families the self-reported delinquency sibling correlations were .32 in brothers, .27 in sisters, and .15 in boy–girl sibling pairs, a value about half that of the same-sex sibling correlations. To some extent, a lower boy–girl correla-

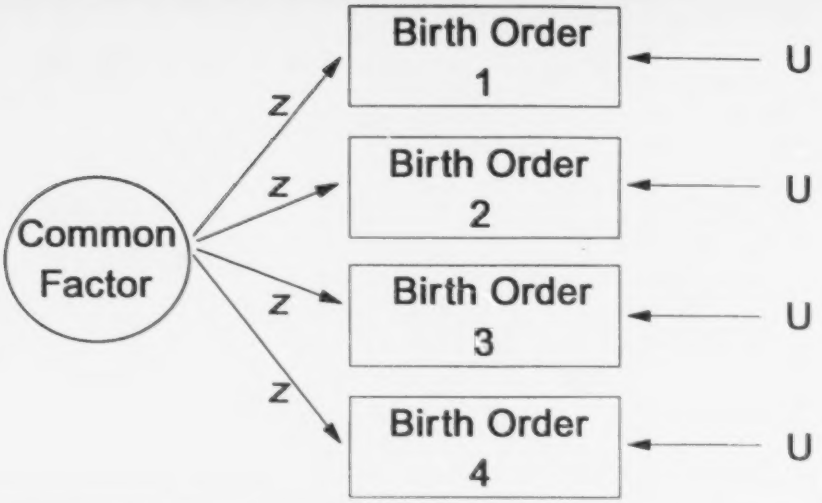

FIGURE 7.1 Common factor model of siblings' delinquency (factor loading z).

tion could result from statistical artifacts. As mentioned earlier, delinquency has a distribution that is skewed in the two sexes, a property that may reduce the size of correlation coefficients. Nevertheless, another interpretation is that familial determinants of delinquency may be sex-specific; hence, they may fail to contribute as much to boy–girl resemblance as they do to same-sex sibling resemblance. With the correlation coefficients mentioned earlier, about 50% (.15/.30) of the etiological influences would be held commonly by males and females. Other data suggest a figure closer to 70% (Rowe, Rodgers, & Meseck-Bushey, 1992).

Composite Genetic Versus Composite Shared Environmental Variation

To distinguish genetic from family environmental influences, a quasi-experimental design is required that compares pairs of relatives who differ from each other in social and/or biological relatedness. In our work on delinquent behavior, we used the twin study research design (Rowe, 1983). The twins were located through high schools and twin clubs in Ohio. The majority of the twins received their questionnaires along with preaddressed return envelopes. The instructions emphasized the importance of completing the forms independently and, if the twins chose to compare responses, not changing a response

because a co-twin had responded differently. The twins' zygosity was diagnosed on the basis of their reports of physical traits (e.g., eye color and height) and appearance similarity. The twins came from a range of social class backgrounds: 20% of their fathers had never completed high school, whereas 27% of the fathers had completed 4 or more years of college. Few twins, however, resided in inner-city areas, and most were Caucasian.

The twin design capitalizes on the greater genetic resemblance of monozygotic (one egg; MZ) twins in comparison to dizygotic (two egg; DZ) twins. If a trait is genetically determined, then reared-together MZ twins should resemble each other in behavior more than reared-together DZ twins. In a study of IQ in 7- to 12-year-old children in the Fels Research Institute's classic study of child development (Rodgers & Rowe, 1987), we used five levels of genetic relatedness: random pairs, cousins, half-siblings, full siblings, and twins. In this case, the rank order of resemblance for a genetically influenced trait should follow the relatives' biological relatedness—from cousins (low) to MZ twins (high).

In this study of IQ variation, we found significant heritability (h^2 = .39) and shared family environmental effects (c^2 = .39, the same estimate as h^2 to two decimal places) that together accounted for the majority of IQ variation (78%). These results are confirmed from a large body of research on IQ showing statistically significant genetic and family environmental components in IQ variation. Nonetheless, the broad research literature has been interpreted as showing that family environmental influences on IQ may be greater in young children than in adulthood (McGue, Bouchard, Iacono, & Lykken, 1993). From a review of recent twin and adoption studies, McGue and his colleagues (1993) concluded that:

"Heritability [of IQ] increases from approximately 50% in preadolescence to approximately 80% in adulthood. In contrast, shared environmental influences appear to decrease from approximately 20% in preadolescence to near 0% in adulthood" (p. 71).

From the standpoint of psychological science, a diminished etiologic role of family influences on IQ with maturity has been unexpected. This finding can be interpreted as "niche picking" (Scarr & McCartney, 1983), a tendency of people with different genotypes to find environmental contexts that reinforce genetic dispositions. As children mature and are exposed to a greater variety of environmental opportunities, they can pick available "niches" more closely matching their genetic dispositions; hence, genetically different family members (e.g., siblings) become less alike, and IQ heritability rises.

In a study of 265 same-sex adolescent twins' delinquency, statistical evidence for both composite variance components was also found (Rowe, 1983). Table 7.2 shows genetic and environmental parameter estimates from a biometrical genetic analysis of the between- and within-twin pair mean squares (i.e., statistical parts of overall twin correlation coefficients). In male twins, these parameter estimates were $h^2 = .34$ and $c^2 = .32$; in females, they were $h^2 = .41$ and $c^2 = .25$. Although the twin sample size was too small to demonstrate the statistical significance of both variance components simultaneously, the variance estimates suggest that both heredity and family-tied environmental influences produce variation in delinquent behavior.

Although few studies of self-reported delinquency are available, there are a number of legitimate twin studies on delinquency. On the basis of their summary of these studies, DiLalla and Gottesman (1989) concluded that twin concordances for MZ twins' delinquency ($M = .87$) were similar to those for DZ twins ($M = .72$), indicating strong family-tied environmental influences on delinquency (and possibly, biased police response to family members). In contrast, adult criminals showed twin concordances to be more consistent with genetic influence (M MZ twins = .51; M DZ twins = .22). Overall, greater evidence exists for some kind of shared family influence on delinquency than on many other traits. In summary, neither studies of IQ nor delinquency can neglect genetic influences—for this reason, a family study research design alone cannot resolve composite influences on these traits. Delinquency is a relatively unique trait in that it yields kinship correlations consistent with shared family environmental influences. Finally, some causes of delinquency may be modified by biological sex.

TABLE 7.2 VARIANCE COMPONENT ESTIMATES FOR SELF-REPORT DELINQUENCY

Group	Heritability h^2	Shared Environment c^2	Nonshared Environment e^2
Male Twins	.34	.32	.34
Female Twins	.41	.25	.33
All Twins	.39	.28	.34

Note. Variance percentages from "Biometrical Genetic Models of Self-Reported Delinquent Behavior: A Twin Study," by D.C. Rowe, 1983, *Behavior Genetics, 13,* Table V, p. 485. Copyright 1986 by Plenum Publishing. Adapted with permission. Variance components for log transformed self-report delinquency scores.

VARIANCE COMPONENTS OF TRAIT COVARIATION

Traits do not occur in isolation; their correlated pattern with other traits can help to define them conceptually and unite different levels of analysis; for instance, the covariance of a behavioral trait with a physiological measure may reveal its biological roots. As was the case for univariate analyses, the covariance of traits can be examined at several levels. First, one can evaluate trait co-occurrence in a family pedigree. In psychiatric research studies, the familial co-occurrence of symptoms is called trait *co-morbidity* but the same conceptual analysis can be applied to any set of behavioral traits. Second, as in the univariate case, the covariation of two traits can be attributed to genetic, shared environmental, and nonshared environmental sources of variation. Third, specific measured variables can be included in multivariate, behavior genetic research designs (Neale & Cardon, 1992).

IQ Covariates

Although we did not explore the covariates of IQ in our research program, other behavior geneticists made effective use of multivariate genetic analyses. In one line of research, the covariation of IQ with elementary information-processing tasks was explored. A recent special issue of the journal *Intelligence* published 11 articles documenting correlations between biological measures and intelligence. Several articles focused on reaction time. These reaction-time tasks are "elementary" in that they require simple recognition decisions or timed responses to letters or numbers, all easily made with few errors. Despite their uncomplicated nature, tasks that tap speed of information processing correlate substantially with IQ (e.g., speed of scanning alphanumeric symbols in short-term memory, or speed of comparing physically different or same word pairs; Baker, Vernon, & Ho, 1991). In Baker et al.'s study (1991), the correlation of reaction times with IQs ranged from –.4 to –.6 (which is so because shorter reaction times are associated with higher IQs). Baker and her colleagues analyzed twin cross-trait correlations, that is, the correlation of reaction time in twin A with IQ in twin B.

Analogous to a univariate correlation, the size of a cross-trait correlation may depend on the twins' genetic relatedness (i.e., MZ or DZ). That is, if the cross-trait correlation in MZ twins is greater

than that in DZ twins, genetic influence can be inferred under the usual assumptions of twin method—genetic additivity and equal (trait-relevant) twin environments. From their twin analyses, Baker and her colleagues (1991) concluded that "the speed-IQ association found here is entirely explained by genetic factors. Genetic correlations were negative and at or near unity" (p. 364). This multivariate genetic work thus reveals that underpinning IQ variation may be genetic variation in the capacity to perform such elementary cognitive operations as memory scanning and stimulus recognition. Further progress along these lines could lead to the identification of specific biological processes held in common by IQ and information-processing measures.

Delinquency Covariates

Delinquency theories propose various correlates of possible significance in the etiology of delinquent behavior. Trait-based theories focus on personality differences between delinquents and nondelinquents. For instance, in the widely read social control theory (Gottfredson & Hirschi, 1990), delinquency is attributed to a persistent trait of low self-control established through early socialization in the family during childhood and resistant to change thereafter. Early family socialization also figures prominently in social learning explanations of crime—through a lack of effective punishment for disobedient behavior (Patterson, DeBaryshe, & Ramsey, 1989). Social learning theory also emphasizes the academic milieu, as persistent school failure may lead to a psychological rejection of conventional social norms and an acceptance of antisocial behaviors. Low grades and lack of ambition for advanced schooling are reliable correlates of delinquent behavior.

These different theories of delinquency, however, rarely consider their explanatory variables from the standpoint of composite variance components detailed earlier. For instance, variation in reports of parental behavior may reflect either shared environmental or genetic variance components. Indeed, a shared environmental variance component would be expected on the basis of social learning theories in which parenting behaviors directly influence children's behavioral outcomes. But suppose the parenting practice is one that is reactive to children's traits—that is, parents may show less affection or use more physical punishment when children are temperamentally

difficult rather than easy going. Given this reversal of causal direction, heritable traits in children should lead to heritable variation in reports of parental behavior. Indeed, twin and adoption studies generally show a genetic component in the variation of ostensibly environmental measures:

> These [twin and adoption] findings suggest that environmental measures often show as much genetic influence as do measures of such behavior as personality. Even though these are very early days in research on the nature of nurture, the results would so far seem to shift the burden of proof to those who continue to assume that environmental measures are free of genetic influence. (Plomin & Bergeman, 1991, p. 384)

The lesson here is that the composite sources of variation in both predictors (independent variables) and in developmental outcomes must be investigated, not assumed.

Using his Ohio twin sample, Rowe (1986) examined the genetic and environmental architecture of delinquency with a set of five behavioral correlates: deceitfulness, anger, impulsivity, value on academic achievement, and parental rejection. Except for the last correlate, these variables can be conceptualized as enduring personality dispositions. Deceitfulness is lying to others (such as parents or friends) to protect oneself from criticism and from the detection of wrongdoing, impulsivity is acting quickly and without forethought, anger is the expression of this emotion in response to provocation, and value on achievement is the importance placed on obtaining good grades in school and on committing oneself to school activities. The last variable, parental rejection, is the perception of parental love and affection toward oneself.

As shown in Table 7.3, these variables all correlate phenotypically with self-reported delinquent behavior. The two personality traits conceptually linked with delinquency yield the strongest associations (deceitfulness, .50; impulsivity, .32). Perceived parental rejection also was associated with a greater number of self-reported acts (r = .27). Although school failure is often mentioned in theories of delinquency, the value on achievement variable was the weakest predictor (r = .19) for these adolescent twins. At issue is whether the covariance among this entire set of variables is attributable to shared environmental, genetic, or nonshared environmental variation.

TABLE 7.3 PSYCHOSOCIAL VARIABLES CORRELATED WITH 630 ADOLESCENTS REPORTS OF DELINQUENT BEHAVIOR

Variable	r
Deceitfulness	.50
Impulsivity	.32
Anger	.28
Parental Rejection	.27
Value on Academic Achievement	.19

Note. Reprinted from "Genetic and Environmental Components of Antisocial Behavior: A Study of 265 Twin Pairs," by D.C. Rowe, 1986, *Criminology, 24*, Table 2, page 523. Copyright 1986 by American Society of Criminology. Adapted with permission.

To answer this question, a statistical model was imposed on the data that defined permissible sources of variation for each trait. This model defined three common factors. Like common factors in an ordinary factor analysis, these factors can explain variation in each variable as well as their intercorrelations. The common factors represented genetic, shared environmental, or nonshared environmental sources of variation and covariation. For example, if several variables load on the common genetic factor, then their intercorrelation may be attributed to pleiotropic effects of genes—namely, the same genes may determine variation in several phenotypes at once. If they load on a common family environmental factor, then the same specific family environmental influences may determine variation and covariation among the set of variables. Hypothetically, parental rejection could lead the twins to devalue school, break the law, and perceive parental hostility in their reports of parental behavior. A common nonshared factor was also permitted to explain variation and covariation. The specific influences associated with this factor operate simultaneously on all variables, but they also make twins dissimilar from each other in behavior.

In addition to the three common factors, the model postulated variable-specific genetic, shared environmental, and nonshared environmental factors. These variable-specific factors could explain variation in a single variable, without contributing to the correlation of that variable with another. The fit of different statistical models was evaluated by a chi square test—holding for the goals of parsimony (fewest factors) and adequate fit (low chi square).

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The best fitting model made one major simplification: Both common and specific shared environmental factors were omitted. Conceptually, many specific sources of environmental variation are naturally associated with the shared environment parameter estimates. To name a few such variables, social class, family religiosity, parental substance use, childrearing styles, family structure (e.g., one vs. two parents), and many other influences studied in social science should operate to make twin siblings alike because of their common experience of the variable's influence. Furthermore, the one family environmental variable actually assessed—perceived parental rejection—did not require a shared environmental factor. Instead, the environmental influence on delinquency and its correlates was primarily nonshared—those influences that operate to make siblings dissimilar to each other in their behavioral traits. These findings emphasize that not all raised-together siblings are equally delinquent; environmental influences must be sought that explain these differences.

Table 7.4 presents the variance contributions of the nonshared environmental factor and genetic common factors. The numbers shown are the proportion of variation in a variable accounted for by the common factor (e.g., 42% of total delinquency variation was attributable to the common factor in males). At least in adolescent males, the nonshared factor explains variation in poor school achievement, delinquency, and perceptions of parental rejection, suggesting common environmental mechanisms inducing the statistical association among them. Possibly, the common influence may be antisocial peer groups befriended by the more antisocial boys. If these boys actively encourage delinquency and poor school achievement, and actively discourage family attachments, then the nonshared factors' contributions to variation may take the pattern of Table 7.4. To the extent that twins have separate peer groups, their peer attachments would operate environmentally as a nonshared influence (and, as discussed later, common friends would have the opposite effect). The pattern of explained variation for a nonshared-environment common factor in females, which emphasizes two temperamental traits, lacks a straightforward interpretation. In another study, work on problem behaviors in late childhood demonstrated that several specific nonshared environmental influences—maternal reading, spanking, and quality of the home environment—were related to twin, sibling, and half-sibling measures of antisocial behavior (Rodgers, Rowe, & Li, 1994).

TABLE 7.4 PROPORTION OF TOTAL TRAIT VARIATION EXPLAINED BY COMMON FACTORS

	Heredity Common Factor		Nonshared Environment Common Factor	
	h_c^2	h_c^2	e_c^2	e_c^2
Delinquency	.42	.32	.24	.05
Deceitfulness	.49	.51	.12	.06
Parental Rejection	.07	.21	.48	.09
Achievement	.06	.09	.47	.00
Anger	.25	.05	.00	.18
Impulsivity	.25	.13	.01	.39

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Turning to the genetic factor in Table 7.4, this factor explained substantial variation in delinquency and deceitfulness. In males, it captured 25% of the variation in both anger and impulsivity. In females, it explained 21% of the variation in parental rejection and 13% of that in impulsivity. However, in both sexes this genetic factor explained little variation in achievement (< .10). Thus, the genetic disposition toward crime appears to reflect more nonintellectual personality traits than academic achievement, which may be more an environmental correlate of delinquency. In summary, covariation in the delinquency domain may be attributed to a combination of genetic and nonshared environmental variation.

Precocious Sexuality and Delinquency

In adolescence, precocious sexual behavior and nonsexual delinquent behaviors are associated statistically. In four studies using data collected from surveys of adolescents, we investigated the familial basis of this association (Rodgers & Rowe, 1988; Rodgers & Rowe, 1990; Rodgers, Rowe, & Harris, 1992; Rowe, Rodgers, Meseck-Bushey, & St. John, 1989). Together, they lead to several general conclusions. First, as in the case of delinquent behavior, precocious sexual behavior is familial; precocious sexuality in an older sibling puts a younger sibling at "risk" (and vice versa). Second, both sexuality and delinquency share some common familial determinants, but they also have

unique determinants. The common determinants can be inferred from a sibling "cross-correlation": Precocious sexuality in one sibling is associated with greater self-reported delinquency in another, and vice versa. A common "trait" affecting both sexual behavior and delinquency was estimated to correlate .33 and .47 in brothers and sisters, respectively (Rowe, Rodgers, & Meseck-Bushey, 1989, 1992). Third, younger siblings were found to be more sexually active at a given age than older siblings had been (Rodgers & Rowe, 1988).

Rodgers and associates (1992) explored various explanations for this last finding by relating different family structures to the mean level of sexual behavior (e.g., birth orders, birth spacing). For instance, younger sisters of older brothers may be more sexually active than younger sisters of older sisters, because brothers' friends may provide potential sexual partners for younger sisters (a peer "opportunity" hypothesis). Although several such influence hypotheses could be tested using family structure information, none received unequivocal support. One interesting coincidence was noted, however. In a study of adolescents in Richmond, VA and Tallahassee, FL, pubertal maturation was actually more rapid for younger siblings; they self-reported they were more physically advanced than their older brothers or sisters had been at the same chronological age. Earlier maturation could explain sexual precocity in younger siblings; although admittedly, the explanatory problem now digresses because one must understand why pubertal maturation would depend on birth order position. After combining these findings, it makes sense to consider early sexual behavior as part of a general syndrome of adolescent deviant behavior with underlying familial causes.

Specific Sources of Variance Components

The discovery of a composite variance component in trait variation leaves many questions unanswered because many developmental processes would be consistent with the presence of a particular variance component. If the component is "genetic variation," then a difficult challenge is to identify which specific genes are involved. If the component is "shared environment," the problem is to identify which possible mechanism of family environmental influence results in trait variation.

Presently, we know that many specific sources that comprise the composite variance components are unknown for IQ and

delinquency. In the case of IQ variation, several nonshared environmental influences can be named that generally lower adult IQ, such as prenatal alcohol exposure and severely premature birth. In the general population, however, these events are relatively rare; they may account for little of the total IQ variation. Recently, we found correlations between several measured indicators of nonshared influence, such as if the mother or caretaker reads to the child, how often the mother takes the child to the museum, the quality of the home environment, or IQ/achievement scores (Rodgers, Rowe, & May, 1994).

Other common, nonshared environmental events that systematically affect IQ are unknown. Similarly, there are hundreds of Mendelian (single gene) disorders in human populations, and many of them affect IQ adversely. But, like fetal alcohol syndrome, they are very rare in most populations; they may account for little of the total IQ variation. Currently, molecular genetic research strategies are being applied to identify specific genes associated with IQ variation in the normal range (IQs 70 to 130; see research methods described in Plomin, 1990). It is too early to gauge the effect of these new research efforts.

Similarly, the specific sources of genetic variation in delinquency have not been identified. One chromosomal abnormality—a double dose of the *Y* chromosome in men (genotype: *XYY*)—has been associated statistically with crime. However, this chromosomal abnormality may be linked indirectly through other phenotypic effects of abnormality, such as lower intelligence (Witkin et al., 1976). Whatever its causal basis, this rare abnormality should account for only a tiny proportion of variation in delinquent or adult criminal behavior. Although molecular genetic methods may be used to scrutinize the composite genetic variation in delinquent behaviors, no large-scale scientific work is being conducted yet, partly because national funding agencies are embroiled in a political controversy over researching a biological basis of crime.

Composite Shared Environment and Delinquency

As mentioned earlier, several studies of delinquent behavior yield composite shared environmental components. In another research project, we examined one hypothesis for the specific source of this composite variation; namely, that it results from peer influence in the family. In nearly all studies of delin-

quency, delinquent behavior in friends and associates is the best statistical predictor of an adolescent's own delinquency. Causal ambiguity is present here: delinquent youths may befriend one another ("birds of a feather"), or a delinquent youth may influence a nondelinquent one. Most researchers, however, regard the relationship as bidirectional—as partly selection, partly influence (Rowe, Woulbroun, & Gulley, 1993).

In the context of the family, the influential "peer" may be a sibling or friends of the sibling. This reasoning leads to the following prediction: Siblings who have contact with one another, or overlapping friendship networks, should have similar delinquent behavior traits than isolated siblings because of a mutual and reinforcing influence of peers' delinquency on one's own behavior. Siblings' friendships would then operate as a "moderator" variable that influences their resemblance for delinquent acts.

We tested this hypothesis in a study of 418 sibling pairs, all of adjacent birth order (Rowe & Gulley, 1992). The siblings were located in Tucson, AZ through a random-dialing procedure. The final Arizona Sibling Relationship Study sample consisted of siblings forming 141 mixed-sex sibling pairs, 135 brother pairs, and 142 sister pairs (a total of 836 individuals in 418 families). Their mean age was 13.5 years. Most of the families were middle class, as few mothers (3%) had failed to complete high school and many (44%) had a college-level education or better. Interviews were computer administered to each sibling independently. Among other questions asked, each child completed a standard self-report delinquency inventory, a measure of emotional closeness to the other sibling, and an item covering the number of the siblings' common friends. About 20% of the brothers and 13% of the sisters reported frequent contact with common friends; thus, only a minority of adolescent siblings had strongly overlapping friendship networks.

Same-sex siblings' mutual closeness and their friendship networks moderated the sibling resemblance in delinquency. Similar moderating effects did not hold for boy–girl sibling pairs (who generally were more emotionally distant than were same-sex siblings). Table 7.5 presents sibling correlations for two levels of overlapping friendship networks. Brothers with common friends resembled each other more in delinquency than did brothers with separate friends (r = .20 vs. .63); similarly, sisters with common friends were more alike than were those without them (r = .33 vs. .65). The significant correlations for siblings with little mutual contact (or mutual liking) suggests that peer effects were

TABLE 7.5 DELINQUENCY CORRELATIONS FOR SIBLING PAIRS WITH SEPARATE AND MUTUAL FRIENDS

	Brothers		Sisters	
Group	*r*	*N* Pair	*r*	*N* Pair
Separate Friends	.20	108	.33	124
Mutual Friends	.66	27	.65	18
All Siblings	.44	135	.37	142

Note. Boy/Girl delinquency correlation was nonsignificant. Adapted from "Sibling Effects on Substance Use and Delinquency," by D.C. Rowe & B.L. Gulley, 1992, *Criminology, 30,* Table 3, p. 227. Copyright 1992 by American Society of Criminology. Reprinted with permission.

not the only source of sibling similarity, a clear alternative was genetic influence via the siblings' resemblance in heritable personality traits (such as impulsivity and anger mentioned earlier). Yet peer influence does add an extra degree of similarity to some pairs, making them markedly alike in their delinquent behavior. Thus a major source of the composite shared environmental influence seen in twin studies may be this peer influence effect (which should be greater for twins than nontwin siblings, given that the former are always the same age). A recent analysis of Danish twins supports this interpretation. A twin contact effect explained part of the variation in delinquency, and estimates of the heritability of delinquency were reduced from those based on the "classic" twin model (Carey, 1992).

Uniting the Threads

This chapter covers a broad spectrum of results from behavior genetic studies of IQ and delinquency, emphasizing work done in our research program. It illustrates some of the methodological possibilities of the sibling research design, such as estimating composite variance components, apportioning covariance to variance components, and seeking specific sources of variance components. In our studies of IQ and in related research, we found that IQ is highly heritable and influenced, beyond childhood, primarily by nonshared environmental influences. Heritable brain functions, as tapped by information-processing tasks, may underlie IQ variation.

In our studies of delinquency, we found that delinquency variation apportioned among all three variance components. Further-

more, the degree of shared environmental influence was exceptionally large. Genetic influence also linked delinquency with temperamental traits (impulsivity); familial influences (either genetic or family environmental) linked it with precocious sexual behavior. Siblings belonging to the same peer network were more alike in delinquent behavior than were siblings who had different friends or who were emotionally distant from each other.

None of these findings, though, constitutes a theory of delinquent behavior. Why did one sibling's delinquency so easily influence that of another? What makes early sexual behavior a familial covariate of delinquency? An imaginative leap, from inductive use of data to deductive theory, is needed to place these findings about delinquency into a broader context.

Moffitt (1993) proposed a theory of delinquency that integrates many of our findings. She believes that adolescent delinquency is actually heterogeneous, composed of a small group of life-course-persistent delinquents who are antisocial at every life stage and a larger group of adolescence-limited delinquents who are basically healthy teenagers mimicking the first group's antisocial behavior. Life-course-persistent delinquents, about 5% of males, exhibit a variety of behavioral problems in childhood. Their childhood traits include lying, hyperactivity, impulsiveness, and aggression. These traits are heritable both in childhood and adulthood; as reported earlier, at least lying (the deceitfulness variable) and impulsivity are *genetic* correlates of delinquency. Because of their personality traits, these children are difficult to socialize; they do not form bonds with others readily and fail in many important life tasks. They embark on an antisocial life course that often continues through adulthood.

Adolescence-limited delinquency exhibits a different set of characteristics, most notably a temporal sequence: Delinquency starts in adolescence and usually ends in the late teens or early 20s. Many males have at least some police contact during adolescence, and most confess in self-report surveys to at least some minor illegal acts. Therefore, the adolescence-limited form of delinquency can be regarded as statistically "normal" in the United States. Furthermore, most adolescence-limited delinquents accept later the normal adult responsibilities of work and parenting. With psychological maturity, they become healthy and productive members of society.

Why do these young people temporarily adopt behaviors that are, at least on the surface, harmful? Moffitt (1993) blamed a temporal gap between physical puberty and adult social and

economic responsibilities in Western societies. The onset of biological maturity brings with it an interest in sex and romantic attachment, a wish for independence from family, and a desire to establish rank against other boys (note that these developmental trends are phylogenically shared with other primates). To many adolescent boys, the life-course-persistent delinquent seems to have achieved some of these markers of adult maturity. As Moffitt observed:

> Life-Course-Persistent youths are able to obtain possessions by theft or vice that are otherwise inaccessible to teens who have no independent incomes (cars, clothes, drugs, entry into "adults-only" leisure settings). Life-Course-Persistent boys are more sexually experienced and have already initiated relations with the opposite-sex. [They] . . . appear relatively free of their families of origin; they seem to go their own way, making their own rules. As evidence that they make their own decisions, they take risks and do dangerous things that parents could not possibly endorse. (p. 25)

Average adolescents may be drawn toward mimicking antisocial behavior because it is a means of achieving adult maturity and removing apron strings of childhood dependence. These adolescents, however, possess the social and intellectual traits that would enable them to adopt conventional adult roles. They shift into these roles as opportunities arise (a job or a marriage) and as the costs of delinquency increase (e.g., penalties for crime increase at age 16 or 18 in many states). In contrast, life-course-persistent delinquents possess traits and learning histories that make this transition more difficult; with options foreclosed, they tend to remain antisocial into adulthood (e.g., through crime, abusive parenting, and other forms of antisocial behavior).

The idea that mimicry drives the spread of such behaviors among adolescents is at the heart of our recently developed theory of social contagion (Rodgers & Rowe, 1993). In past work, we borrowed models from epidemiology to explain the spread of smoking (Rowe, Chassin, Presson, Edwards, & Sherman, 1992), drinking (Rowe & Rodgers, 1991), and sexuality (Rodgers & Rowe, 1993; Rowe, Rodgers, & Meseck-Bushey, 1989) through social networks of adolescents. Although these models must be adapted (to accommodate nonbinary behaviors), our social contagion theory can apply to mildly deviant behaviors. Specifically,

the life-course-persistent delinquent adolescents "seed" the system and then "spread" delinquency through social contagion. In the language of epidemiological models, normal adolescents will eventually "recover" from such influence. The small group of persistent delinquents remain "infected" with delinquency for life. These ideas of the spread of deviance similar to our previous work on social contagion can be incorporated into mathematical models.

In our findings on siblings' friendships, the mimicry of delinquent behavior is apparent in the sibling effect: When one sibling (or that child's friends) is delinquent, then there is a high likelihood of behavioral imitation. Those siblings who contact one another easily influence one another; those siblings belonging to different peer groups lack this source of behavioral resemblance and consequently may correlate more weakly on delinquency than the high-contact siblings. In this particular sample, the majority of the delinquent siblings were probably of the adolescent-limited type. Nevertheless, some sibling resemblance on delinquency may arise from a presence in the sample of life-course-persistent delinquents, who share predisposing traits via genetic inheritance. In addition, other heritable traits may be shared by siblings who never exhibit delinquent behavior (e.g., extreme shyness, timidity, and depression). These heritable traits would also induce a sibling resemblance.

The within-person correlation between delinquency and precocious sexuality is also understandable in these terms: Physically mature adolescents seek their adult identities through initiating sexual behavior. The life-course-persistent delinquents move in this direction very quickly; their sexuality is precocious.[1] The adolescent-limited delinquents mimic them. The familial covariance between early sexuality and delinquency probably has multiple bases. First, it may be partly a function of siblings' similar rates of pubertal maturation. When both siblings mature late, they would be unlikely to engage in delinquent behavior. Second, it may also arise from those heritable traits related to delinquency and nondelinquency. Third, the social mimicry of delinquent acts also would make siblings alike, at least for those pairs in which the siblings were mutually close.

In the context of socialization research, these conclusions have one anomalous component—blame for antisocial behavior

[1] Early sexual behavior and childbearing would also maintain genes for the life-course-persistents' pathological traits in the population.

is not placed on parents. In general, there is little evidence of parent–child transmission of behavioral traits; family environment–child developmental outcomes are often mediated genetically (Rowe, 1994). Family heredity molds children's behavioral dispositions, which then express themselves against the constraints of culture and immediate environmental influences.

As mentioned earlier, the story of the Wideman brothers gives a nonfictional account of how heredity and environment may interact. Why were John and Robby Wideman's lives so different? As their personalities differed from an early age, their genes for such traits as impulsivity may have differed. They also had different nonshared environmental experiences. The brothers were separated in age by about nine years. Drugs were less available to John in the late 1950s and early 1960s than to Robby in the late 1960s. Robby was exposed to a street drug "culture" that had developed during this decade. Furthermore, Robby's friends were unlike John's, as John observed, "My father called them [Robby's friends] thugs. Robby and his little thugs" (Wideman, 1984, p. 10). Personality traits, immediate nonshared environmental conditions (e.g., drugs, local peer groups), and broader cultural conditions (e.g., social attitudes toward drugs) all differentiated the two brothers. Within this complex interaction of heredity and nonshared environment, we may yet discover why John and Robby Wideman led such different lives—one an outstanding career as a writer and professor of English; the other, a life diminished by crime and imprisonment—and also why siblings in general differ so greatly in behavior.

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